

# Retraction: Mechanosensitive channel candidate MCA2 is involved in touch-induced root responses in *Arabidopsis*

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**Keywords:** mechanosensitive channel, *Arabidopsis*, root, skewing, waving, calcium, touch response, mechanical stress

## A retraction of the Original Research Article:

**Mechanosensitive channel candidate MCA2 is involved in touch-induced root responses in *Arabidopsis***

by Nakano, M., Samejima, R., and Iida, H. (2014). *Front. Plant Sci.* 5:421. doi: 10.3389/fpls.2014.00421

The authors and the journal wish to retract the 21 Aug 2014 article cited above in light of new experimental evidence.

Following the publication of the study, we performed a DNA microarray analysis to detect genes with expression levels that were specifically lower in the *mca2*-null mutant than in the Col-0 wild type, and found that the expression level of the *AXR4* gene (At1G54990), which encodes a protein required for the subcellular localization of the auxin influx carrier AUX1 (Dharmasiri et al., 2006), was significantly lower in the *mca2*-null mutant. To confirm this finding, we then performed a semi-quantitative reverse transcription-PCR analysis using the primers *axr4*-f1 and *axr4*-Cr1 (Figure 1A), and found that the RT-PCR product was detectable in some *mca2*-null seedlings at wild-type levels but not in other *mca2*-null seedlings at all. This result suggested that some *mca2*-null seedlings have a certain lesion in the *AXR4* locus, and a PCR-based genomic deletion analysis (Figures 1B,C) followed by DNA sequencing confirmed this speculation. Our conclusion is that most of the *mca2*-null seedlings used in the study presented in the above paper had a homozygous 2592-bp deletion that started from the intron between exons 1 and 2 of the *AXR4* gene and reached the intron between exons 1 and 2 of the adjacent gene *AT1G55000* (Figure 1A). Therefore, the phenotypes presented in the above paper may be ascribable to the *axr4* mutation, the *at1g55000* mutation, or both or even triple mutations, but not to the *mca2*-null mutation. The *AT1G55000* gene encodes the peptidoglycan-binding LysM domain-containing protein involved in a macromolecule catabolic process in the cell wall<sup>1</sup>.

Our phenotypic study revealed that none of the *mca2*-null *AXR4*<sup>+</sup> *AT1G55000*<sup>+</sup> seedlings showed all the abnormal phenotypes reported in the above paper, regarding the skewing, waving, and bending responses of the root. In contrast, the seedlings of the *axr4-1* (Ws-2 background) and *axr4-2* (Col background) single mutants obtained from the Arabidopsis Biological Resource Center (ABRC germplasm names CS8018 and CS8019, respectively)

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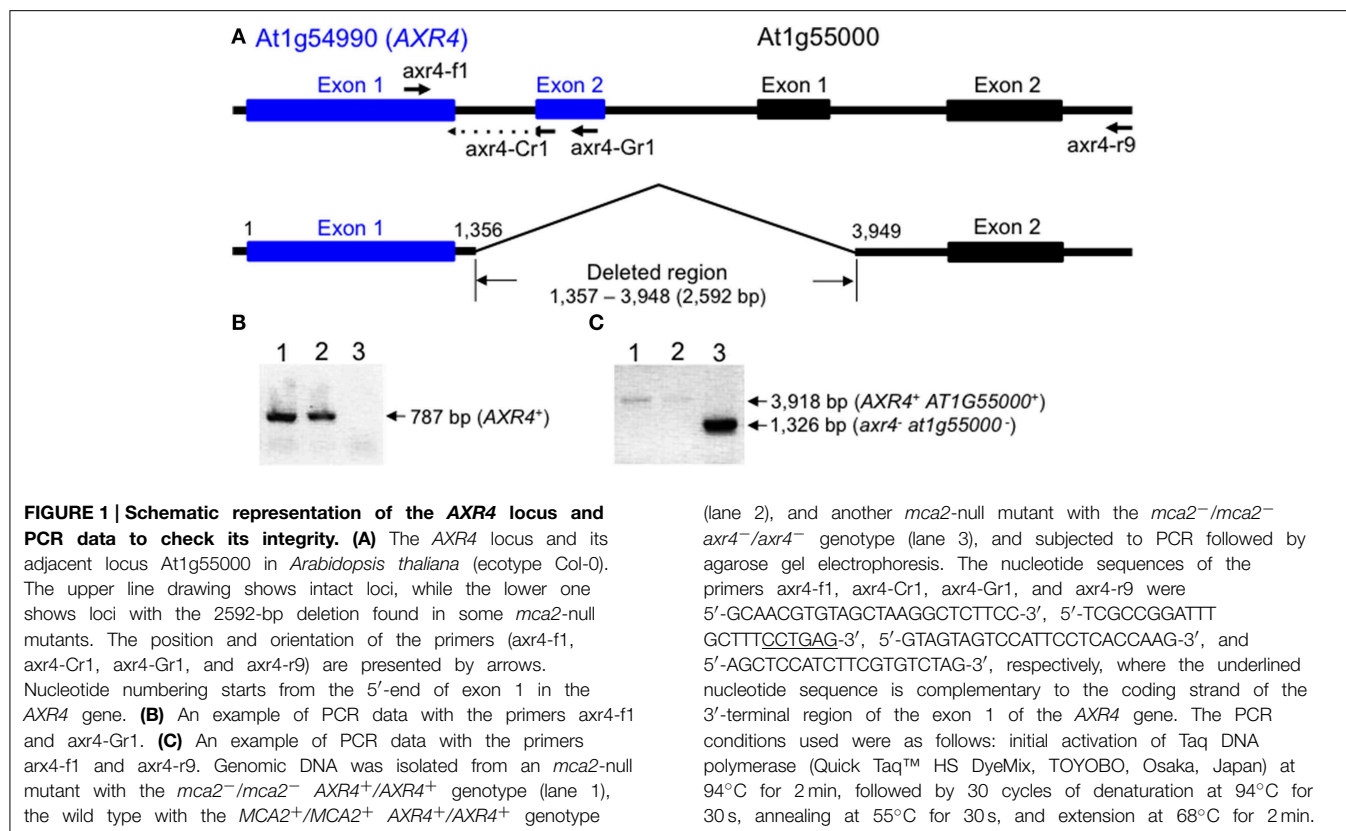
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<sup>1</sup>Database of The Arabidopsis Information Resource (TAIR) at <http://www.arabidopsis.org/servlets/TairObject?type=locus&name=AT1G55000>



showed the same abnormal phenotypes as those described in the above paper. We also confirmed that the abnormal phenotypes for the skewing, waving, and bending responses of the *mca2*-null *axr4 at1g55000* triple mutant were identical to those of the *axr4-1* and *axr4-2* single mutants. These findings clearly demonstrated that the abnormal phenotypes described in the above paper were ascribed solely to the mutation in the *AXR4* gene.

An important question is why did some of our *mca2*-null germplasms have the *axr4*<sup>-</sup>/*axr4*<sup>-</sup> *at1g55000*<sup>-</sup>/*at1g55000*<sup>-</sup> allele? We had never used *axr4* mutants in our laboratory before the above paper was published. We speculated that some of the seeds of the *mca2*-null mutant (germplasm name: SALK\_129208) obtained from the ABRC 12 years ago were heterozygous for the *AXR4* and *AT1G55000* loci (i.e., *AXR4*<sup>+</sup>/*axr4*<sup>-</sup> *AT1G55000*<sup>+</sup>/*at1g55000*<sup>-</sup>), and multiple self-pollinations performed by us to maintain seed viability produced seed

stocks with *axr4*<sup>-</sup>/*axr4*<sup>-</sup> *at1g55000*<sup>-</sup>/*at1g55000*<sup>-</sup> as well as *AXR4*<sup>+</sup>/*AXR4*<sup>+</sup> *AT1G55000*<sup>+</sup>/*AT1G55000*<sup>+</sup> and *AXR4*<sup>+</sup>/*axr4*<sup>-</sup> *AT1G55000*<sup>+</sup>/*at1g55000*<sup>-</sup>, although all of the 20 seeds of the *mca2*-null mutant (germplasm name: SALK\_129208C), which were newly obtained from the ABRC and tested, had the genotype of *AXR4*<sup>+</sup>/*AXR4*<sup>+</sup> *AT1G55000*<sup>+</sup>/*AT1G55000*<sup>+</sup>. As for the *MCA2* locus, 19 out of the 20 seeds had the *mca2*<sup>-</sup>/*mca2*<sup>-</sup> genotype and one had the *MCA2*<sup>+</sup>/*mca2*<sup>-</sup> genotype. The *mca1*-null *mca2*-null double mutant and *mca2*/*MCA2* complementation lines used in the above paper were *AXR4*<sup>+</sup>/*AXR4*<sup>+</sup> *AT1G55000*<sup>+</sup>/*AT1G55000*<sup>+</sup>. Furthermore, the *mca2*-null single and *mca1*-null *mca2*-null lines used in our previous study (Yamanaka et al., 2010) were also *AXR4*<sup>+</sup>/*AXR4*<sup>+</sup> *AT1G55000*<sup>+</sup>/*AT1G55000*<sup>+</sup>.

We deeply regret any scientific misconceptions that have been caused by the above paper and apologize to the scientific community for any adverse consequences.

## References

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**Conflict of Interest Statement:** The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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